1. A method for lessening or preventing non-pulmonary ischemia-reperfusion injury in a mammal, comprising the steps of:

(a) identifying a mammal that has ischemia-reperfusion or is at risk for developing ischemia-reperfusion in a non-pulmonary tissue; and

(b) causing the mammal to inhale a therapeutically effective amount of gaseous nitric oxide sufficient to diminish the ability of the mammal's leukocytes or platelets to become activated in a manner that contributes to an inflammatory process at the site of the ischemia-reperfusion in the non-pulmonary tissue, thereby lessening or preventing non-pulmonary ischemia-reperfusion injury in the mammal.

2. The method of claim 1, further comprising the step of administering to the mammal a therapeutically effective amount of a second compound that potentiates the therapeutic effect of gaseous nitric oxide.

3. The method of claim 2, wherein the second compound is selected from the group consisting of a phosphodiesterase inhibitor and superoxide dismutase.

4. The method of claim 3, wherein the phosphodiesterase inhibitor is selected from the group consisting of 2-o-propoxyphenyl-8-azapurin-6-one, dipyridamole, theophylline, sildenafil, and 1,3-dimethyl-6-(2-propoxy-5-methanesulphonylamidophenyl)-pyrazolo[3,4-D]pyrimidin-4-(5H)-one.

5. The method of claim 2, wherein the second compound is selected from the group consisting of aspirin,

ticlopidine, streptokinase, urokinase, t-PA and analogs thereof, heparin, and hirudin and analogs thereof.

6. The method of claim 1, wherein the injury is caused by surgery.

7. The method of claim 6, wherein the surgery is transplantation surgery.

8. The method of claim 7, wherein the transplantation surgery is kidney transplantation surgery or heart transplantation surgery.

9. The method of claim 6, wherein the surgery is heart bypass surgery.

10. The method of claim 1, wherein the injury is caused by a vascular interventional procedure.

11. The method of claim 10, wherein the vascular interventional procedure is angioplasty.

12. The method of claim 11, wherein the angioplasty includes the use of a laser, balloon, or stent.

13. The method of claim 11, wherein the angioplasty is an atherectomy.

14. The method of claim 10, wherein the vascular interventional procedure is percutaneous transluminal coronary angioplasty.

15. The method of claim 1, wherein the injury is caused by thrombolysis.

16. The method of claim 1, wherein the injury is caused by a stroke.

17. The method of claim 1, wherein the injury occurs in the kidney.

18. The method of claim 1, wherein the injury occurs in the heart.

19. The method of claim 1, wherein the injury occurs in the brain.

20. The method of claim 1, wherein the injury occurs spontaneously.

21. The method of claim 1, wherein the therapeutically effective amount of nitric oxide is administered to the mammal at a predetermined concentration range.

22. The method of claim 21, wherein the concentration range is 0.1 ppm to 300 ppm.

23. The method of claim 1, wherein the nitric oxide is inhaled continuously for an extended period.

24. The method of claim 1, wherein the nitric oxide is inhaled intermittently for an extended period.

25. The method of claim 1, wherein the mammal is a human.

26. The method of claim 1, wherein the amount of gaseous nitric oxide is sufficient to diminish the ability of platelets to become activated in a manner that contributes to the inflammation process at the site of the ischemia-reperfusion.

27. A method for decreasing or preventing non-pulmonary inflammation in a mammal, comprising the steps of:

(a) identifying a mammal which has existing inflammation or is at risk for developing inflammation in a non-pulmonary tissue;

(b) causing the mammal to inhale a therapeutically effective amount of gaseous nitric oxide sufficient to diminish the ability of the mammal's leukocytes or platelets to become activated in a manner that contributes to an inflammation process in the non-pulmonary tissue, thereby decreasing or preventing non-pulmonary inflammation in the mammal; and

(c) immediately before, during, or after the inhalation of nitric oxide by the mammal, administering to the mammal a therapeutically effective amount of a second compound that potentiates the therapeutic effect of gaseous nitric oxide.

28. The method of claim 27, wherein the non-pulmonary inflammation is arthritis, myocarditis, encephalitis, transplant rejection, systemic lupus erythematosis, gout, dermatitis, inflammatory bowel disease, hepatitis, or thyroiditis.

29. The method of claim 27, wherein the second compound is selected from the group consisting of a phosphodiesterase inhibitor and superoxide dismutase.

30. The method of claim 29, wherein the phosphodiesterase inhibitor is selected from the group consisting of 2-o-propoxyphenyl-8-azapurin-6-one, dipyridamole, theophylline and 1,3-dimethyl-6-(2-propoxy-5-methanesulphonylamidophenyl)-pyrazolo[3,4-D]pyrimidin-4-(5H)-one.

31. The method of claim 27, wherein the second compound is selected from the group consisting of a non-steroidal anti-inflammatory agent, a glucocorticoid, and a cytotoxic agent.

32. The method of claim 27, wherein the nitric oxide is inhaled in a predetermined concentration range.

33. The method of claim 32, wherein the concentration range is 0.1 ppm to 300 ppm.

34. The method of claim 27, wherein the nitric oxide is inhaled continuously for an extended period.

35. The method of claim 27, wherein the nitric oxide is inhaled intermittently for an extended period.

36. The method of claim 27, wherein the mammal is a human.

37. The method of claim 27, wherein the amount of gaseous nitric oxide is sufficient to diminish the ability

of platelets to become activated in a manner that contributes to the inflammation process.

38. The method of claim 1, wherein the injury is caused by trauma.

39. The method of claim 1, wherein the injury is caused by temporary hypotension.